Food and Chemical Toxicology 105 (2017) 456–474

Contents lists available at ScienceDirect

Food and Chemical Toxicology

journal homepage: www.elsevier.com/locate/foodchemtox

Review

Potential contribution of insecticide exposure and development of obesity and type 2 diabetes

Xiao Xiao [a], John M. Clark [b], Yeonhwa Park [a, *]

[a] Department of Food Science, University of Massachusetts, Amherst, MA 01003, USA
[b] Department of Veterinary and Animal Sciences, University of Massachusetts, Amherst 01003, MA, USA

ARTICLE INFO

Article history:
Received 13 January 2017
Received in revised form
13 April 2017
Accepted 3 May 2017
Available online 6 May 2017

Keywords:
Insecticide
Obesity
Glucose metabolism
Type 2 diabetes

ABSTRACT

The introduction of insecticides has greatly improved agricultural productivity and human nutrition; however, the wide use of insecticides has also sparked growing concern over their health impacts. Increased rate of cancers, neurodegenerative disorders, reproductive dysfunction, birth defects, respiratory diseases, cardiovascular diseases and aging have been linked with insecticide exposure. Meanwhile, a growing body of evidence is suggesting that exposure to insecticides can also potentiate the risk of obesity and type 2 diabetes. This review summarizes the relationship between insecticide exposure and development of obesity and type 2 diabetes using epidemiological and rodent animal studies, including potential mechanisms. The evidence as a whole suggests that exposure to insecticides is linked to increased risk of obesity and type 2 diabetes.

© 2017 Elsevier Ltd. All rights reserved.

Contents

* Corresponding author. Department of Food Science, University of Massachusetts, Amherst 102 Holdsworth Way, Amherst, MA 01003, USA.
E-mail address: ypark@foodsci.umass.edu (Y. Park).

http://dx.doi.org/10.1016/j.fct.2017.05.003
0278-6915/© 2017 Elsevier Ltd. All rights reserved.

1. Introduction

The prevalence of obesity among adults in the United States more than doubled since the 1960s, increasing from 13.4 in 1962 to 35.7 percent in 2010 (NCHS, 2012). As obesity is a well known risk factor for other diseases, especially type 2 diabetes, the incidences of type 2 diabetes are also rising (ADA, 2013; CDC, 2014; WHO, 2013). The current epidemic of obesity and type 2 diabetes cannot be fully explained by changes in societal, genetic, behavioral, or dietary habits of individuals, suggesting unknown factors contribute to these disease outbreaks.

The challenge of growing more food to feed the world's expanding population has driven need to control pest insects, which are harmful to crop yield. Application of insecticides are considered to be a major contributing factor for increased agricultural productivity in the 20th century (van Emden and Peakall, 1996). In addition to its role in agriculture, insecticides are widely used in industry, households, and military to control insect pests that are disease vectors, suggesting its essential role in human life (Sparks, 2013). However, insecticides are one of the major environmental contaminations and the extensive use of insecticides has caused wide public concern over their potential risk of inducing human chronic diseases, including obesity and diabetes (Hectors et al., 2011; Karami-Mohajeri and Abdollahi, 2011; Kuo et al., 2013; Lee et al., 2014a; Rezg et al., 2010b). Among insecticides, organochlorines and organophosphorus are the most investigated insecticides linked with obesity and/or type 2 diabetes in humans and rodents (Karami-Mohajeri and Abdollahi, 2011). Although limited, other types of insecticides, such as carbamates, pyrethroids, and neonicotinoids, are associated with development of obesity and/or type 2 diabetes as well (Montgomery et al., 2008; Narendra et al., 2008; Saldana et al., 2007; Sun et al., 2016b; Wang et al., 2011b). More recently, *in vitro* and *in vivo* studies reported that pyrethroids, a neonicotinoid, and a phenylpyrazole (fipronil), all were involved in potentiated adipogenesis and/or altered glucose responsiveness, as representative of obesity and type 2 diabetes, respectively (Kim et al., 2013, 2014a; Park et al., 2013; Shen et al., 2017; Sun et al., 2016a, 2016b). Moreover, reports of interaction between dietary fat and insecticides on these markers, further begs the question of the role of insecticides and health. Thus, this review focuses on summarizing the current reports on insecticide exposure and development of obesity and type 2 diabetes, including suggested mechanisms, to further expand our current understanding.

2. Methods

Bibliographic databases including PubMed and Web of Science were searched for the keywords ‘insecticide’ and ‘obesity’; ‘insecticide’ and ‘diabetes’; ‘insecticide’ and ‘body weight’; ‘insecticide’ and ‘glucose metabolism’; and ‘insecticide’ and ‘lipid metabolism’. Data were collected from 1966 to December 2016. From the initial search of PubMed and Web of Science, each article was reviewed to evaluate title and abstract content, and to eliminate duplicates and those were not related to our purposes. A total of 111 articles were selected and their full texts were reviewed including 52 human studies and 59 animal studies. Our exclusion criteria were (1) publications containing no original research (reviews, editorials, or non-research letters); (2) studies not carried out in humans, mice, or rats; or (3) human studies without providing information on markers of obesity or diabetes as described below. We collected the following data for human studies: authors, journal, year of publication, country, insecticide, study design, study population, changes in body weight and/or body mass index, waist circumference, diabetes risk (increased blood glucose levels, insulin resistance, or gestational diabetes), lipid markers (triglycerides and/or cholesterols), and other critical comments. Variables extracted from animal studies include: authors, journal, year of publication, species, treatment methods (dose, route, and duration), sex, body weight change, major markers of glucose homeostasis (glucose, insulin, or insulin resistance) and lipid metabolism (triglycerides and/or cholesterol), and other metabolic markers involved in glucose and lipid metabolisms (e.g., blood leptin, other lipids, glycogen content, markers of adipogenesis, gluconeogenesis, glycogenolysis, glycolysis, and/or inflammation). The findings are presented in Tables.

3. Insecticide classification and action

Insecticides can be classified based primarily on their chemical structures and mode of actions. Major classes of insecticides include organochlorines, organophosphorus, carbamates, pyrethroids, and neonicotinoids. A few examples are shown in Table 1.

Organochlorine insecticides can be divided into dichlorodiphenyltrichloroethane (DDT)-type and chlorinated alicyclic-type (cyclodienes) based on their distinctive mechanisms of action. DDT-type insecticides (such as DDT, DDE, and DDD) are known to inhibit the closing of voltage-sensitive sodium channel (VSSC) in neurons, resulting in repetitive firing of action potentials, while chlorinated alicyclic-type insecticides (e.g. aldrin, dieldrin, heptachlor, and endosulfan) bind to γ-aminobutyric acid (GABA) chloride ionophore complex, which inhibits chloride influx into the nerve (Coats, 1990; Karami-Mohajeri and Abdollahi, 2011). Even though most countries have not used organochlorines for the last several decades, due to extremely stable chemical characteristics, DDE (as a major metabolite of DDT) can currently be found in human serum, adipose tissue, and many foods (Karami-Mohajeri and Abdollahi, 2011; USDA, 2014).

Organophosphorus insecticides are irreversible inhibitors of cholinesterases, including acetyl cholinesterase, resulting in hyperstimulation of cholinergic nerves (e.g. muscarinic and nicotinic acetylcholine receptor) (Abou-Donia, 2003). As the largest insecticide class in the world in 1980s, organophosphorus insecticides occupied 71% of world insecticides market in 1987; however, the use of organophosphorus insecticides dropped to around 52% in 1999 and to 13% in 2013 due to its environmental persistence and mammalian toxicity (Casida and Quistad, 2004; Nauen and Bretschneider, 2002; Sparks, 2013).

Carbamates, which account for 6% of global insecticides (Sparks, 2013), have the similar mechanism of action with organophosphorus insecticides, but their neurotoxic effects are relatively more moderate than organophosphorus because the inhibition of acetyl cholinesterase is reversible and carbamates are known to be rapidly metabolized by human and animals (Karami-Mohajeri and Abdollahi, 2011; Risher et al., 1987).

Pyrethroids are structural analogs to naturally occurring insecticide, pyrethrin, found in *Chrysanthemum* flower heads. Pyrethroids can cause over excitation of the neuron by delaying the closing of VSSC, producing an effect similar to, but more pronounced than, DDT due to its better sodium channel binding capacity (Davies et al., 2007; Soderlund et al., 2002). By 2013, pyrethroids accounted for approximately 16% of the global insecticide market (Sparks and Nauen, 2015).

Neonicotinoids are a relatively new family of insecticides with structural resemblance to nicotine (Casida and Durkin, 2013). Acting on nicotinic acetylcholine receptors, neonicotinoids can stimulate these receptors at low doses, while blocking these receptors at high doses, leading to paralysis and death (Gervais et al., 2012). Neonicotinoids have become the fastest growing class of insecticides, representing ~27% of the global insecticide market in

Download English Version:

https://daneshyari.com/en/article/5560166

Download Persian Version:

https://daneshyari.com/article/5560166

Daneshyari.com